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Issue Date: 27 March 2007

In the Matter of:

Mrs. W.W.,[1]
Widow of Mr. J.R.W.,
Claimant

Case No.: 2005 BLA 5785

v.

CONTINENTAL MINING CO./
WEST VIRGINIA CWP FUND,
Employer/Insurer

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS
Party in Interest

Appearances: Ms. Sandra M. Fogel, Attorney
For the Claimant

Mr. Martin E. Hall, Attorney
For the Employer

Before: Richard T. Stansell-Gamm
Administrative Law Judge

DECISION AND ORDER –
AWARD OF SURVIVOR CLAIM

This matter involves a claim filed by Mrs. W.W. for survivor benefits under the Black Lung Benefits Act, Title 30, United States Code, Sections 901 to 945 ("the Act"), as implemented by 20 C.F.R. Parts 718 and 725. Benefits are awarded to persons who are totally disabled within the meaning of the Act due to pneumoconiosis, or to survivors of persons who died due to pneumoconiosis. Pneumoconiosis is a dust disease of the lung arising from coal mine employment and is commonly known as "black lung" disease.

[1]Chief Administrative Law Judge John Vittone has directed that I substitute initials for the names of the Claimant and all family members. Any comments or concerns regarding this mandated practice should be directed to Chief Administrative Law Judge John Vittone, 800 K Street, Suite 400N, Washington, D.C. 20001.

Procedural Background

Mr. J.R.W.'s Claim
DX 1[2]

Mr. J.R.W. filed a claim for benefits under the Act on August 14, 2001. A claims examiner at the Department of Labor ("DOL") determined that Mr. W. was entitled to benefits on May 22, 2003. The copy of the Proposed Decision and Order that was sent to Continental Mining Co. was refused delivery and returned to DOL. On June 5, 2003, the West Virginia Coal Workers' Pneumoconiosis Fund ("West Virginia CWP Fund") objected to DOL's award and requested a formal hearing before the Office of Administrative Law Judges ("OALJ"). On June 30, 2003, the West Virginia CWP Fund notified DOL that it initiated benefit payments. The District Director sent the case file to OALJ on November 26, 2003. Mr. W. passed away on March 11, 2004, and Mrs. W. pursued the claim as his representative. Administrative Law Judge Daniel Leland held a hearing on August 24, 2004 in Charleston, West Virginia. On December 6, 2004, Judge Leland awarded black lung disability benefits to Mr. W. posthumously. The Virginia CWP Fund did not appeal.

Mrs. W.W.'s Claim

After Mr. W. passed away, and while his miner claim was still pending, Mrs. W. filed a claim for survivor benefits under the Act on March 24, 2004 (DX 3). The District Director awarded survivor benefits on January 19, 2005 (DX 25). On January 26, 2005, the West Virginia CWP Fund objected to DOL's award, requested a formal hearing before OALJ, and notified DOL that it initiated benefit payments (DX 26). The District Director sent the case file to OALJ on April 19, 2005 (DX 31).

After a Notice of Hearing dated October 4, 2005 (ALJ I), I set a hearing date of February 15, 2006 for this case in Charleston, West Virginia. Prior to the scheduled hearing, based on a request from Claimant's counsel, and with the concurrence of Employer's counsel, I agreed to render a decision on the record, DX 1 to DX 34. Subsequently, the parties submitted additional evidence for my consideration. In response to the evidentiary issues raised by those submissions, I issued Preliminary Evidentiary Determinations on March 7, 2006 (ALJ II). On July 17, 2006, I received Claimant's motion to strike certain deposition testimony. I received the Employer's response on July 21, 2006.

Preliminary Issues

Collateral Estoppel

In light of a recent decision by the U.S. Court of Appeals for the Fourth Circuit, I have decided to make a *sua sponte* application of collateral estoppel. *Sua sponte* application of collateral estoppel is within the bounds of judicial discretion because "if a court is on notice that

[2]The following notations appear in this decision to identify the exhibits and references: DX – Director exhibit; CX – Claimant exhibit; EX – Employer exhibit; ALJ – Administrative Law Judge exhibit; and, TR – Transcript.

it has previously decided the issue presented, the court may dismiss the action *sua sponte*, even though the defense has not been raised; this result is fully consistent with the policies underlying res judicata, in that it is not based solely on the defendant's interest in avoiding the burdens of twice defending a suit, but is also based on the avoidance of unnecessary judicial waste." *Arizona v. California*, 530 U.S. 392, 412 (2000) (quoting *United States v. Sioux Nation*, 448 U.S. 371, 432 (1980) (Rehnquist, J., dissenting) (citations omitted)).

In November 2006, the U.S. Court of Appeals for the Fourth Circuit handed down its decision in *Collins v. Pond Creek Mining Co.,* No. 05-1832 (4th Cir. Nov. 8, 2006) and applied the principle of nonmutual offensive collateral estoppel[3] in a black lung survivor claim. In *Collins*, a miner was awarded black lung benefits in 1988 and the Employer did not appeal that award. After his death, his widow sought black lung survivor benefits, but she was denied compensation because the ALJ found that her husband did not have pneumoconiosis. The Benefits Review Board ("BRB") upheld the ALJ's decision. Before the Fourth Circuit, the widow argued that the doctrine of nonmutual collateral estoppel precluded the re-adjudication of the issue of whether her husband had pneumoconiosis related to his coal mine employment because it was previously litigated and given a final determination. The Fourth Circuit concluded that after "an issue is actually and necessarily determined by a court of competent jurisdiction," collateral estoppel applies to subsequent suits involving a different cause of action but at least one of the parties from the earlier suit. *Collins*, *Id*. p.5 (quoting *Montana v. United States*, 440 U.S. 147, 153 (1979)). Administrative decisions, like those of the District Director or OALJ, "are to be accorded the same collateral estoppel effect they would receive if made by a court." *Collins*, p.6 (citing *Jones v. SEC*, 115 F.3d 1173, 1178 (4th Cir. 1997)). The Fourth Circuit applied collateral estoppel to find that a survivor can prevent re-litigation of certain issues when the living miner's claim was granted and the employer did not appeal.

The BRB has found only one exception to the application of collateral estoppel in a survivor claim – the existence of autopsy evidence in the survivor claim. In *Villain v. Zeigler Coal Co.*, BRB No. 00-0451 BLA (Jan. 29, 2001) (unpub.), the BRB found that collateral estoppel was appropriate in a survivor's claim because without an autopsy, "an exception does not apply."[4]

Although Ms. W. survivor claim contains autopsy evidence, I find the exception to the application of collateral estoppel in Mrs. W.'s survivor claim does not apply. As noted in the procedural summary, Mr. W. passed away prior the ALJ's evidentiary hearing. As a result, during the adjudication of Mr. W.'s miner claim, both parties presented autopsy/pathology evidence relating to the presence of coal workers' pneumoconiosis in his lung which was fully considered by Judge Leland. Consequently, while both claims in *Collins* only involved chest x-

[3]As noted by the Fourth Circuit, "[c]ollateral estoppel is 'offensive' when a plaintiff seeks to 'foreclose the defendant from litigating an issue the defendant has previously litigated unsuccessfully,' and it is 'nonmutual' when the party seeking to rely on the earlier ruling was not a party to the earlier proceeding and is not in privity with a party." *Collins*, p.6 (quoting *In re Microsoft Corp. Antitrust Litig.*, 355 F.3d 322, 326 (4th Cir. 2004)).

[4]*See also Zeigler Coal Co. v. Director, OWCP*, 213 F.3d 332 (7th Cir. 2002) ("there is no point in re-adjudicating the question whether a given miner had pneumoconiosis unless it is possible to adduce highly reliable evidence—which as a practical matter means autopsy results").

ray and medical opinion evidence, the court's rationale for collateral estoppel directly applies in Mrs. W.'s survivor claim because the same three types of evidence – chest x-ray, autopsy/pathology, medical opinion – were presented in both Mr. W.'s miner claim and Mrs. W.'s survivor's claim[5] on the same issue – whether Mr. W. had coal workers' pneumoconiosis. During the adjudication of that issue, while finding the preponderance of the autopsy evidence to be inconclusive, Judge Leland concluded Mr. W. had coal workers' pneumoconiosis based on the preponderance of the radiographic evidence and probative medical opinion. When the Employer/Insurer did not appeal,[6] Judge Leland's decision awarding black lung disability benefits became final. At that time, Judge Leland's determination that Mr. W. had coal workers' pneumoconiosis also became final, and through the application of nonmutual offensive collateral estoppel, it is now legally established in Mrs. W.'s survivor claim.

Evidentiary Issues

Dr. Rasmussen's December 2001 Medical Opinion
(DX 1)

Despite the extensive amount of relevant medical evidence that may be contained in earlier DOL-sponsored medical evaluations of a deceased miner, and even though miner's prior claims and the associated medical evidence are automatically considered part of the record in his subsequent black lung disability claim under 20 C.F.R. § 725.309(d)(1) and not subject to the evidentiary limits under 20 C.F.R. § 725.414, the BRB rejected a similar automatic inclusion of prior black lung disability claims into a record of the related survivor claim. *Keener v. Peerless Eagle Coal Co.*, ___ B.L.R. ___, BRB No. 05-1008 BLA (Jan. 30, 2007) (en banc), the BRB stated that medical evidence from a miner's claim is not automatically admissible in the related survivor's claim. Instead, the survivor must specifically designate medical evidence from the deceased miner's prior claims. Additionally, such evidence is also subject to the evidence limitations under 20 C.F.R. § 725.414.

In support of her survivor claim, Ms. W. designated the medical opinions of Dr. Cohen (CX 1) and Dr. Green (CX 2) as her two permissible case-in-chief medical opinions under 20 C.F.R. § 725.414(a)(3)(i). Additionally, she designated Dr. Rasmussen's December 2001 assessment as a relevant medical report that qualifies for the "good cause" exception to the evidence restrictions. She also asserted that since DOL sponsored the examination, its admission should not be charged as one of the Claimant's admissible medical reports. However, if relevancy were the threshold for the "good cause" exception, the evidence restrictions imposed by DOL would have no effect. In regards to DOL sponsoring Dr. Rasmussen's pulmonary examination, 20 C.F.R. § 725.406(b) indicates that a DOL examination "shall not be counted as evidence submitted by the miner under § 725.414." Notably, while that provision clearly applies

[5]Both parties in Mrs. W.'s survivor claim presented several additional medical opinions. However, those assessments were based on the same underlying objective medical evidence, particularly the gross autopsy findings and the tissue pathology samples, that were previously evaluated and presented to Judge Leland.

[6]I note that since Mrs. W. filed her survivor claim in March 2004, the Employer/Insurer had ample notice prior to both the August 2004 evidentiary hearing in Mr. W.'s miner claim and Judge Leland's December 2004 award of black lung disability benefits of their additional potential liability for survivor benefits.

in a miner's black lung disability claim, the regulation contains no similar provision for a survivor claim and the BRB in *Keener* did not carve out an exception to its evidence designation mandate in a survivor claim for the DOL sponsored examination. Accordingly, Dr. Rasmussen's opinion from his December 2004 examination is not admissible either for good cause or as the DOL sponsored examination.[7]

Dr. Zaldivar's October 2003 Medical Opinion
(DX 1)

Claimant also seeks admission of Dr. Zaldivar's October 2003 medical opinion under the "good cause" exception. The Claimant argues that the good cause exception should apply because Dr. Zaldivar's 2003 report was "the most comprehensive pulmonary evaluation after the 2001 examination." The Claimant contends that the reviewing physicians should have the most complete picture of Mr. W.'s health when they render their opinion. As stated above, while I acknowledge the good sense behind both arguments, consider relevancy is an insufficient basis for invoking the good cause exception to the evidentiary restrictions. As a result, Dr. Zaldivar's medical opinion is not admissible.[8]

Dr. Cohen's Medical Opinion
(CX 1)

The inadmissibility of the opinions of Dr. Rasmussen and Dr. Zaldivar poses an evidentiary problem for Dr. Cohen's medical opinion because under 20 C.F.R. §§ 725.414(a)(2)(i) and 3(i) "any chest X-ray interpretation, pulmonary function test results, blood gas studies . . . and physician opinions that appear in a medical report must each be admissible" under the regulations. In *Harris v. Old Ben Coal Co.,* 23 B.L.R. 1-98 (2006) (en banc), when confronted with a medical opinion that contained evidence not admitted into the formal record, the BRB indicated that an ALJ may: a) exclude the report, b) redact the objectionable content, c) require a revised report, or d) consider the physician's reliance on the inadmissible evidence in deciding the probative value of the report. In this case, I will apply a combination of the second and fourth options. I will redact any mention by Dr. Cohen of the medical opinions of Dr. Rasmussen and Dr. Zaldivar. And, concerning probative value, I find Dr. Cohen's review of these two inadmissible opinions has little adverse effect because Dr. Cohen did not rely on their specific conclusions. Instead, he rendered an independent decision based on his own assessment of the objective medical evidence.

[7]Nevertheless, based on Claimant's designations, and due to compliance with 20 C.F.R. § 725.414(a)(2)(i), the chest x-ray, pulmonary function study, and arterial blood gas study from the December 2001 examination are admissible.

[8]Again, the objective medical evidence from Dr. Zaldivar's October 2003 pulmonary evaluation has been admitted under 20 C.F.R. § 725.414(a)(2)(i).

Dr. Green's Medical Opinion
(CX 2)

In addition to reviewing medical records from Mr. W.'s miner claim, including the inadmissible opinions of Dr. Rasmussen and Dr. Zaldivar, Dr. Green also referenced a March 21, 2001 report by Dr. Samuel V. Spagnolo, which is not part of the record in this claim. Returning to the *Harris* considerations, redacting is fairly easy since Dr. Green did not mention specific opinions from the other physicians. As to probative value, Dr. Green's principal findings regarding Mr. W.'s medical history and death remain probative since he addressed those issues based on the admitted medical tests, medical treatment records, and autopsy reports.

Employer's Additional Evidentiary Submissions

In addition to EX 1,[9] the Employer submitted the following reports, which I now admit into evidence: interpretation of the October 8, 2001 chest x-ray by Dr. Jerome Wiot (EX 2); interpretation of the January 29, 2003 chest x-ray by Dr. George Zaldivar (EX 3); medical report of Dr. Andrew Ghio (EX 4); medical report of Dr. Thomas Jarboe (EX 5); deposition transcripts of Dr. Jarboe (EX 6) and Dr. Ghio (EX 7); and interpretations of the October 8, 2001 chest x-ray by Dr. William Scott and Dr. Paul Wheeler (EX 8).

Depositions

In a Motion to Strike, counsel for the Claimant requests that I disregard portions of the depositions of Dr. Ghio and Dr. Jarboe because they refer to inadmissible medical information in excess of what is permitted as evidence for the Employer. Specifically, the Claimant argues that Dr. Ghio and Dr. Jarboe referred to the medical reports of Dr. Green and Dr. Cohen, and that this acts as impermissible "rebuttal" evidence. The Employer responds that the Claimant did not raise these concerns during the depositions so they are waived, the reports of Dr. Green and Dr. Cohen are in the record so Dr. Ghio and Dr. Jarboe are free to discuss them, the depositions are not rebuttal evidence, and if I strike portions of the depositions I should also strike the portions of the reports by Dr. Green and Dr. Cohen because they also cited other physicians' medical reports.

In assessing this motion, two regulatory provisions are helpful. Section 725.414(c) states that "[a] physician who prepared a medical report admitted under this section may testify with respect to the claim at any formal hearing . . . or by deposition." Additionally, § 725.414(d) states that a physician "may testify as to any other medical evidence of record, but shall not be permitted to testify as to any medical evidence relevant to the miner's condition that is not admissible." Because the reports of Dr. Green and Dr. Cohen are admitted to the record in this claim, Dr. Ghio and Dr. Jarboe are free to discuss them. As a result, I deny the Claimant's motion to strike.

[9]In the Preliminary Evidentiary Determinations (ALJ II), I admitted as EX 1 Dr. Crouch's examination of the autopsy slides as the Employer's one autopsy report under 20 C.F.R. § 725.414(a)(3)(i). The Benefits Review Board recently confirmed that an employer may submit an autopsy slide review as an autopsy report under 20 C.F.R. § 725.414(a)(3)(i). *Keener v. Peerless Eagle Coal Co.*, ___ B.L.R. ___, BRB No. 05-1008 BLA (Jan. 30, 2007) (en banc).

Summary

In light of my evidentiary determinations, my decision in this case is based on the documents admitted into evidence: DX 1 (other than the medical opinions of Dr. Rasmussen and Dr. Zaldivar) to DX 34, CX 1 to CX 2, and EX 1 to EX 8.

ISSUES

1. Whether Mrs. W. is an eligible survivor.

2. Whether Mr. W.'s death was due to coal workers' pneumoconiosis.

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Coal Miner's Background

Born on September 3, 1937, Mr. J.R.W. married Mrs. W.W. on January 18, 1971. Mr. W. worked underground in the coal mines in 1971, working with roof bolters, cutting machines, and operating a miner until 1982. Between 1985 and 1993, Mr. W. worked off and on operating a roof bolter and doing utility work underground in the mines. After at least 20 years of coal mine employment, Mr. W. stopped working in the mines when they closed down in 1993. Mr. W. smoked half of a pack per day until approximately 1972. Mr. W. passed away on March 11, 2004. (DX 1; TR, p.11)

Survivor Claim

To receive survivor benefits under the Act, and the implementing regulations, 20 C.F.R. § 718.205(a), a claimant must prove by a preponderance of the evidence several facts. First, the claimant must establish eligibility as a survivor. A surviving spouse may be considered eligible for benefits under the Act if she was married to, and living with, the coal miner at the time of his death, and has not remarried.[10]

Next, the claimant must prove the coal miner had pneumoconiosis.[11] "Pneumoconiosis" is defined as a chronic dust disease arising out of coal mine employment. The regulatory definitions include both clinical pneumoconiosis (the diseases recognized by the medical community as pneumoconiosis) and legal pneumoconiosis (defined by regulation as any chronic lung disease arising out of coal mine employment).[12] The regulation further indicates that a lung

[10]20 C.F.R. § 718.4 indicates that the definitions in 20 C.F.R. § 725.101 are applicable. 20 C.F.R. § 725.101, in turn, refers to the term "survivor" as used in Subpart B of Part 725. 20 C.F.R. § 725.214 then sets out the spousal relationship requirements and 20 C.F.R. § 725.215 describes the dependency rules. According to § 725.214(a) the spousal relationship exists if the relationship is a valid marriage under state law. Under § 725.215(a), a spouse is deemed dependent if she was residing with the miner at the time of his death.

[11]20 C.F.R. § 718.205(a)(1); *see Trumbo v. Reading Anthracite Co*., 17 B.L.R. 1-85 (1993).

[12]20 C.F.R. §§ 718.201(a) (1) and (2).

disease arising out of coal mine employment includes "any chronic pulmonary disease or respiratory or pulmonary impairment significantly related to, or substantially aggravated by, dust exposure in coal mine employment."[13] As courts have noted, under the Act, the legal definition of pneumoconiosis is much broader than medical pneumoconiosis. *Kline v. Director, OWCP*, 877 F.2d 1175 (3d Cir. 1989).

Once a determination has been made that a miner had pneumoconiosis, it must be determined whether the coal miner's pneumoconiosis arose, at least in part, out of coal mine employment.[14] If a miner who was suffering from pneumoconiosis was employed for ten years or more in one or more coal mines, there is a rebuttable presumption that pneumoconiosis arose out of such employment.[15] Otherwise, the claimant must provide competent evidence to establish the relationship between pneumoconiosis and coal mine employment.[16]

Finally, the surviving spouse has to demonstrate the coal miner's death was due to pneumoconiosis.[17]

In summary, a survivor claim filed after January 1, 1982 must meet four primary elements for entitlement. The claimant bears the burden of establishing these elements by a preponderance of the evidence. If the claimant fails to prove any one of the requisite elements, the survivor claim for benefits must be denied. *Gee v. W. G. Moore and Sons*, 9 B.L.R. 1-4 (1986); *Roberts v. Bethlehem Mines Corp*., 8 B.L.R. 1-211 (1985). The four elements are: (1) the claimant is an eligible survivor of the deceased miner, (2) the coal miner suffered from pneumoconiosis, (3) the coal miner's pneumoconiosis arose out of coal mine employment, and (4) the coal miner's death was due to coal workers' pneumoconiosis.

Again, based on nonmutual offensive collateral estoppel and Judge Leland's final determination, I find that Mr. W. had coal workers' pneumoconiosis.

Issue # 1 – Eligible Survivor

Based on Mrs. W.'s statement of eligibility on her claim for black lung benefits (DX 3), a copy of Mr. and Mrs. W.'s marriage certificate (DX 9), and Mrs. W.'s testimony at the August 24, 2004 hearing (TR, p.9), and absent evidence to the contrary, I find that Mrs. W. is an eligible survivor under the Act.

[13]20 C.F.R. § 718(b).

[14]20 C.F.R. §§ 718.203(a) and 205(a)(2).

[15]20 C.F.R. § 718.203(b).

[16]20 C.F.R. § 718.203(c).

[17]20 C.F.R. § 718.205(a)(3).

Issue # 2 – Death Due to Pneumoconiosis

Through the application of nonmutual offensive collateral estoppel and Judge Leland's final determination and as an eligible survivor, Mrs. W. has established the first three elements of entitlement(eligible survivor, presence of pneumoconiosis, and pneumoconiosis arising out of coal mine employment). As a result, Mrs. W. may receive survivor benefits if the preponderance of the evidence in the record establishes that her husband's death was due to coal workers' pneumoconiosis. For a survivor claim filed on or after January 1, 1982, DOL regulations provide four means to establish that a coal miner's death was due to coal workers' pneumoconiosis:[18]

1. The miner had complicated pneumoconiosis;[19]

2. Death was caused by pneumoconiosis;

3. Death was caused by complications of pneumoconiosis; or,

4. Pneumoconiosis was a substantially contributing cause or factor leading to the miner's death. Notably, pneumoconiosis is deemed to be a substantially contributing cause of a miner's death if it hastens the miner's death.[20]

Additionally, a survivor may not receive benefits if the coal miner's "death was caused by a traumatic injury or the principal cause of death was a medical condition not related to pneumoconiosis, unless evidence establishes that pneumoconiosis was a substantially contributing cause of death."[21]

As a result, Mrs. W. must prove death due to pneumoconiosis through one of the four means noted above. Prior to assessing each factor, I will summarize the medical evidence consisting of chest x-rays, pulmonary tests, treatment reports, and post-mortem medical reports.

Chest X-Rays

Date of x-ray	Exhibit	Physician	Interpretation
June 10, 2001	DX 13	Dr. Bailes	Fluffiness, perhaps a sarcoid type picture with some interstitial changes.
June 13, 2001[22]	DX 13	Dr. Salvador	Diffuse fluffy densities on both lungs, could be secondary to congestion but fluffy pneumonic

[18]20 C.F.R. §§ 718.205(c)(1)-(3), 718.304.

[19]According to 20 C.F.R. § 718.304, if a miner had complicated pneumoconiosis, an irrebuttable presumption exists that his death was due to pneumoconiosis.

[20]20 C.F.R. § 718.205(c)(5).

[21]20 C.F.R. § 718.205(c)(4).

[22]As noted in Dr. Salvador's treatment notes from Mr. W.'s March 10, 2003 treatment visit.

			infiltrate cannot be excluded.
October 8, 2001	DX 15	Dr. Patel, B, BCR[23]	Positive for simple pneumoconiosis, profusion category 2/2,[24] type t/u opacities,[25] and honeycomb lung.
(same)	DX 15	Dr. Ahmed, BCR, B	Positive for simple pneumoconiosis, profusion category 2/2, type t/q opacities, right peripheral pleural thickening.
(same)	DX 1	Dr. Cappiello, BCR, B	Positive for simple pneumoconiosis, profusion category 2/3, type q/t opacities, coalescent opacities both lower lung zones, right chest wall pleural thickening, honeycombing
(same)	EX 2	Dr. Wiot, BCR, B	Positive for pneumoconiosis, profusion category 2/2, type t/q opacities. "Compatible" with coal workers' pneumoconiosis, but more likely IPF or other disease involving lung bases.
(same)	EX 8	Dr. Scott, BCR, B	Negative for pneumoconiosis. Decreased lung volumes, possible interstitial fibrosis, possible [unreadable] IP, advise high resolution CT, cannot rule out edema, atypical pneumoconiosis.
(same)	EX 8	Dr. Wheeler, BCR, B	Negative for pneumoconiosis. Ill-defined interstitial infiltrate or fibrosis lower lungs and lower lateral "rul" involving pleura. Check for autoimmune disease and usual interstitial pneumonitis. No silicosis or CWP, no benign asbestos-related pleural plaques which should be present with asbestosis.
January 29, 2003	DX 1	Dr. Cappiello, BCR, B	Positive for simple pneumoconiosis, profusion category 2/3, type q/t opacities, coalescent opacities left lower lung zone, right chest wall pleural thickening, honeycombing in lower lung zones.

[23]The following designations apply: B – B reader, and BCR – Board Certified Radiologist. These designations indicate qualifications a person may posses to interpret x-ray film. A "B Reader" has demonstrated proficiency in assessing and classifying chest x-ray evidence for pneumoconiosis by successful completion of an examination. A "Board Certified Radiologist" has been certified, after four years of study and examination, as proficient in interpreting x-ray films of all kinds including images of the lungs. *See also* 20 C.F.R. § 718.202(a)(1)(ii).

[24]The profusion (quantity) of the opacities (opaque spots) throughout the lungs is measured by four categories: 0 = small opacities are absent or so few they do not reach a category 1; 1 = small opacities definitely present but few in number; 2 = small opacities numerous but normal lung markings are still visible; and, 3 = small opacities very numerous and normal lung markings are usually partly or totally obscured. An interpretation of category 1, 2, or 3 means there are opacities in the lung which may be used as evidence of pneumoconiosis. If the interpretation is 0, then the assessment is not evidence of pneumoconiosis. A physician will usually list the interpretation with two digits. The first digit is the final assessment; the second digit represents the category that the doctor also seriously considered. For example, a reading of 1/2 means the doctor's final determination is category 1 opacities but he considered placing the interpretation in category 2. Additionally, according to 20 C.F.R. § 718.102(b), a profusion reading of 0/1 does not constitute evidence of pneumoconiosis.

[25]There are two general categories of small opacities defined by their shape: rounded and irregular. Within those categories the opacities are further defined by size. The round opacities are: type p (less than 1.5 millimeter (mm) in diameter), type q (1.5 to 3.0 mm), and type r (3.0 to 10.0 mm). The irregular opacities are: type s (less than 1.5 mm), type t (1.5 to 3.0 mm) and type u (3.0 to 10.0 mm). JOHN CRAFTON & ANDREW DOUGLAS, RESPIRATORY DISEASES 581 (3d ed. 1981).

(same)	EX 3	Dr. Zaldivar, B	Negative for pneumoconiosis. Honeycombing in left lung, pleural thickening in the interlobar fissure of mediastinum, and (unreadable) nodular infiltrate bilaterally lower & mid zones.
September 9, 2003	DX 14	Dr. Older	History of black lung. Lung volumes markedly reduced with pulmonary fibrosis bilaterally, reticular-nodular interstitial prominence most pronounced at bases. Nodular density in upper lobes, may be related to interstitial lung disease or mass lesion.
December 25, 2003	DX 13	Dr. Setliff	Prominent interstitial markings bilaterally, suspected CHF, evidence of extensive bilateral chronic lung disease.
January 7, 2004	DX 13	Dr. Maki	Significant bilateral pulmonary infiltrate. Prominent bronchovascular and interstitial markings. Poor inspiratory effort. Pneumonia cannot be completely excluded.
January 9, 2004	DX 13	Dr. Nicholas	Marked reticular nodular densities bilaterally, no segmental consolidations evident, no pleural effusion, chronic interstitial fibrosis with questionable superimposed pneumonitis.
February 11, 2004	DX 13	Dr. Salvador	Increased interstitial markings.
February 13, 2004	DX 13	Dr. Nicholas	Chronic interstitial fibrosis evident. No consolidations or effusions. Heart is not enlarged.
February 14, 2004	DX 13	Dr. Rose	Patchy increase in the interstitial and alveolar lung markings in the right lung and majority of the left, most pronounced in left mid-to-lower zones. May reflect pulmonary fibrosis. No definite acute pneumonia but associated pneumonia process cannot be excluded on this x-ray. Probable pulmonary fibrosis. Heart is normal.
March 8, 2004	DX 14	Dr. Robbins	Patient with advanced, progressive coal workers' pneumoconiosis. Chest x-ray showed bilateral interstitial disease.
March 9, 2004	DX 13	Dr. Nicholas	Severe chronic change, no segmental infiltrations or consolidations, no effusions evident.
March 11, 2004	DX 14	Dr. Brown	Complete opacification of both lungs with a background of reticular nodular pattern bilaterally. Decreased lung volumes consistent with interstitial fibrosis. The opacification may be edema, infiltrate, or collapse.

Pulmonary Function Tests

Exhibit	Date Doctor	Age Height	FEV^1 pre[26] post[27]	FVC pre post	MVV pre post	% FEV^1/FVC pre post	Qualified[28] pre post
DX 13	October 4, 2001 Dr. Durham[29]	64 69”	2.16 2.04	2.75 2.62	62 52	78.5% 77.9%	No[30] No
DX 15	October 8, 2001 Dr. Rasmussen	64 67”	2.47 --	2.83 --	92 --	87.3% --	No[31]
DX 1	January 29, 2003 Dr. Zaldivar	65 68”	1.86 1.89	2.16 2.11	-- --	86.1% 89.6%	No[32] No

Arterial Blood Gas Studies

Exhibit	Date / Doctor	pCO_2 (rest) pCO_2 (exercise)	pO_2 (rest) pO_2 (exercise)	Qualified[33]
DX 13	June 10, 2001 Dr. Bailes	36	73	No[34]
DX 15	October 8, 2001 Dr. Rasmussen	33 37	77 46	No[35] Yes[36]
DX 1	January 29, 2003 Dr. Zaldivar	37 35	71 45	No Yes[37]

[26]Test result before administration of a bronchodilator.

[27]Test result following administration of a bronchodilator.

[28]Under 20 C.F.R. § 718.204(b)(2)(i), to qualify for total disability based on pulmonary function tests, for a miner’s age and height, the FEV1 must be equal to or less than the value in Appendix B, Table B1 of 20 C.F.R. § 718, and either the FVC has to be equal or less than the value in Table B3, or the MVV has to be equal or less than the value in Table B5, or the ratio FEV1/FVC has to be equal to or less than 55%. Table B1 sets out the qualifying FEV1 values, but the values do not extend past age 71. However, the qualifying FEV1 values decrease as age increases, so I will infer qualifying results based on the age 71 values.

[29]This is admitted as part of Mr. W.’s treatment records.

[30]The qualifying FEV1 value for age 64 and 69” is 1.90.

[31]The qualifying FEV1 value for age 64 and 67” is 1.75.

[32]The qualifying FEV1 value for age 65 and 68” is 1.82.

[33]To qualify for Federal Black Lung Disability benefits at a coal miner’s given pCO_2 level, the value of the coal miner’s pO_2 must be equal to or less than corresponding pO_2 value listed in the Blood Gas Tables in Appendix C for 20 C.F.R. § 718.

[34]For the pCO_2 of 36, the qualifying pO_2 is 64 or less.

[35]For the pCO_2 of 33, the qualifying pO_2 is 67 or less.

[36]For the pCO_2 of 37, the qualifying pO_2 is 63 or less

[37]For the pCO_2 of 35, the qualifying pO_2 is 65 or less.

DX 13	December 23, 2003 Dr. Worthington	39.5	72	No[38]
DX 13	January 7, 2004 Dr. Wantz	44.3	22.4 (on oxygen)	Yes
DX 13	February 11, 2004 Dr. Greenberg	35.6	71.7 (on oxygen)	No
DX 13	February 11, 2004 Dr. Salvador	40.1	48.4	Yes
DX 13	February 17, 2004 (unknown)	40.0	56.6 (on oxygen)	Yes
DX 14	February 18, 2004 (unknown)	43	64	No
DX 14	February 2004[39] (unknown)	40	36 (on oxygen)	Yes
DX 13	March 9, 2004 Dr. Wantz	37.5	24.2 (on oxygen)	Yes[40]
DX 13	March 9, 2004 Dr. Wantz	37.8	27.6 (on oxygen)	Yes
DX 13	March 9, 2004 Dr. Wantz	40.2	57.9 (on oxygen)	Yes
DX 13	March 9, 2004 Dr. Wantz	47.2	73.4 (on oxygen)	No
DX 13	March 10, 2004 Dr. Wantz	44.9	59.3 (on oxygen)	Yes
DX 14	March 11, 2004 (unknown)	54	31	Yes

Treatment Records
(DX 12-14)

On June 10, 2001, Mr. W. entered the Summersville Memorial Hospital emergency room with shortness of breath. His breath sounds were clear. He was discharged the same day. On July 20, 2001 Mr. W. was given an echocardiogram, which revealed normal cardiac chambers, mild aortic sclerosis and mitral annular calcification without regurgitation or evidence of pulmonary hypertension. On October 4, 2001, Mr. W. underwent a pulmonary function test, which was interpreted by Dr. Durham as showing a moderate restrictive process with decremental changes on bronchodilators.

On March 10, 2003, Dr. Salvador saw Mr. W. for shortness of breath. Mr. W. mined coal for 25 years and smoked cigarettes for 7 1/2 years but he quit 30 years ago. An earlier visit to Dr. Durham revealed an obstructive and restrictive lung pathology. Mr. W. had coarse rales in the lower third of his lungs. Dr. Salvador prescribed nebulizers and constant oxygen therapy.

[38]For the pCO_2 of 40-49, the qualifying pO_2 is 60 or less.

[39]As noted in Dr. Cavangna's treatment note dated February 20, 2004.

[40]For the pCO_2 of 38, the qualifying pO_2 is 62 or less.

On March 23, 2003, Dr. Salvador saw Mr. W. for shortness of breath. Mr. W. was doing well on home oxygen therapy, but he had coarse rales in both lower lungs. Dr. Salvador noted chronic obstructive pulmonary disease ("COPD"), restrictive process, and history of coal mine exposure.

On May 9, 2003, Dr. Enelow treated Mr. W. for dyspnea. Mr. W. worked in the coal mines and was told numerous times that he had coal workers' pneumoconiosis. He smoked a pack a day for about 30 years, but quit almost 30 years ago. Upon physical exam, Mr. W.'s chest was remarkable for "rather dense" crackles at his bases, going about half way up his chest. There were no significant wheezes or rhonchi. Quiet heart sounds. Dr. Enelow reviewed Mr. W.'s CT scan and chest x-rays,[41] and found that Mr. W. had "rather dense" fibrotic changes, patchy but "rather diffusely," largely subpleural, but sparing few areas of the lung. Increased severity in the basilar regions. There were small nodes evident in the hilar region, and calcification was evident on some cuts. A pulmonary function test done on May 9, 2003 showed reduced lung capacity compared to his "prior numbers."[42] Dr. Enelow's impression was that Mr. W. was a 65 year old gentleman in remarkably good health except for "rather severe fibrotic lung disease of uncertain etiology." The degree of his lung impairment suggested that his disease was quite advanced. Based on that, Dr. Enelow thought that an exact etiology determination was not worthwhile. His "guess" was that it was "a form of occupational lung disease, not clearly classic coal worker's pneumoconiosis, but probably an element of silicosis as well." Although Mr. W. seemed to benefit from nebulizers, he did not have any evidence of airflow obstruction on May 8, 2003. Mr. W. could suffer from idiopathic pulmonary fibrosis, which was a possibility worth considering in Dr. Enelow's opinion because it would give Mr. W. access to treatment options. Dr. Enelow did not think Mr. W. had interstitial pulmonary fibrosis with symptoms going back 20 years. Given that Mr. W. was physiologically 5 to 10 years younger than his chronological age, he might be a candidate for a lung transplant.

Dr. Salvador saw Mr. W. on June 23, 2003. Mr. W. was on home oxygen therapy and was recently diagnosed with end-stage pulmonary fibrosis. Mr. W. had fine rales in both lungs.

In September 2003, underwent another evaluation. Mr. W. had advanced coal workers' pneumoconiosis. He worked in the coal mines for many years until 8 years ago, and smoked until he quit 20 years ago. He was recently placed on oxygen, but he is now very limited in his activities. After a battery of tests, Dr. Robbins recommended Mr. W. for a lung transplant on September 9, 2003.

A CT scan on September 10, 2003 revealed pleural thickening at the left base, and diffuse areas of intralobular septal thickening, subpleural cysts with focal areas of traction bronchiectasis most notably in the periphery. These findings were most prominent in the bases. There were a few areas of ground-glass opacity at the bases, and no pulmonary masses. There were multiple enlarged mediastinal lymph nodes. Dr. Petruzzi's pulmonary impression was: 1) extensive mediastinal lymphadenopathy, which may be related to the diffuse interstitial lung disease, and

[41]The dates of these tests are unknown.

[42]The dates and results of these tests are not included in the record.

lymphoma should be considered, and 2) diffuse areas or peripheral pulmonary fibrosis most prominent at the bases, and focal areas of ground-glass opacity at the lung bases suggesting active inflammation.

Dr. Salvador examined Mr. W. on September 23, 2003. Mr. W. was on home oxygen, and had fine rales in both lung bases. Dr. Salvador examined Mr. W. on December 16, 2003. Mr. W. had COPD with pulmonary fibrosis, was on home oxygen, and Dr. Salvador heard no rales, rhonchi, or wheezes.

Dr. Salvador examined Mr. W. on December 23, 2003 for a cold and lung problems, including shortness of breath. Mr. W. had decreased breath sounds, and Dr. Salvador referred him to the emergency room. Mr. W. was admitted to the hospital on December 23, 2003 and was discharged on December 25, 2003.[43] Dr. Salvador saw Mr. W. for a follow-up on January 2, 2004 and noted decreased, clear breath sounds and some expiratory coarseness with cough. Dr. Salvador's assessment was black lung/pneumoconiosis and some CHF (congestive heart failure).

Mr. W. entered the Summersville Memorial Hospital on January 6, 2004 for shortness of breath. He smoked cigarettes for about 12 years. His January 7, 2004 chest x-ray showed significant bilateral pulmonary prominent bronchovascular and interstitial markings, and pneumonia could not be excluded. A follow-up chest x-ray on January 9, 2004 showed chronic interstitial fibrosis with a questionable superimposed pneumonia. The echocardiogram on January 7, 2004 was "abnormal." He did not wheeze, but he continued to have diffuse bilateral crackles which went along with his interstitial fibrosis. He was diagnosed with clinical pneumonia, severe COPD/interstitial fibrosis, and pneumoconiosis. He was discharged on January 13, 2004.

Dr. Salvador saw Mr. W. for a follow-up visit on January 20, 2004. Mr. W. had COPD and was using home oxygen. Mr. W. had fine rales on either side, and no wheezes or rhonchi. Dr. Salvador's assessment was that Mr. W.'s pneumonia was resolved.

Mr. W. reentered the hospital on February 11, 2004 following a routine check-up with Dr. Salvador. At Summersville, Dr. Greenberg noted that Mr. W. experienced shortness of breath upon walking 5 feet. Mr. W. was a coal miner for 20 years and smoked from age 18 until his mid 30s. Mr. W. had diffuse wheezes and crackles, with "okay air movement." His chest x-rays showed severe pulmonary fibrosis with no significant evidence of pneumonia. Pneumonia could not be ruled out, however, because of the extent of the fibrosis. Mr. W. continued to "desaturate" upon minimal effort. The echocardiogram was "borderline." Dr. Greenberg diagnosed COPD exacerbation and severe fibrosis. The February 15, 2004 echocardiogram was "abnormal."

Mr. W. was transferred from Summersville to the University of Virginia Hospital on February 17, 2004. Dr. Cavangna noted that his medical history included pulmonary fibrosis and advanced coal workers' pneumoconiosis. Mr. W. was on nebulizers, Advair, and oxygen at home. Upon physical exam, Mr. W. had good air movement, with diffuse soft crackles especially at the bases, rare wheezes, and significantly prolonged expiratory phase. Mr. W. was

[43]These treatment records are not in the record, but other treatment notes refer to this hospital admission.

admitted to rule out possible pneumonia. There was no clear evidence of pneumonia on the CT scan done at another hospital as interpreted by Dr. Robbins. A February 17, 2004 CT scan showed severe underlying pulmonary fibrosis, scarring, and emphysematous changes, and Dr. Maki noted that it would be difficult to exclude pneumonia. The physicians felt that his symptoms were more consistent with the progression of his pulmonary fibrosis than with pneumonia. Although his need for continuous oxygen increased, he was discharged on February 20, 2004.

Dr. Robbins treated Mr. W. on March 8, 2004 and found that Mr. W. had advanced coal workers' pneumoconiosis. Mr. W. was on oxygen and had bilateral crackles in his lungs and the chest x-ray showed bilateral interstitial disease. Mr. W. was awaiting a lung transplant.

On March 9, 2004, Mr. W. was admitted to the Summersville Memorial Hospital for shortness of breath and blue fingertips. His medical history included severe fibrosis/pneumoconiosis. He worked in the coal mines for 20 years, and smoked from age 18 until his mid-30s, quitting in 1970. Mr. W.'s respiratory effort was moderate to severe, and he could not speak when he arrived, but this improved. He had distant breath sounds and faint crackles bilaterally. The echocardiogram was "abnormal." Dr. Wantz' impression was that Mr. W. had respiratory failure, severe COPD/pneumoconiosis/interstitial fibrosis, and possible pneumonia. Mr. W. stabilized on oxygen, and was transferred to the University of Virginia Hospital on March 10, 2004. There, Mr. W. had an "abnormal" echocardiogram, Dr. Koenig noted that Mr. W. declined aggressive measures to prolong his life, and he passed away on March 11, 2004.

Post-Mortem Medical Reports

(Note: the following summary of the post-mortem medical reports includes specific autopsy findings, submitted to support Mrs. W.'s survivor claim. While respecting the dignity and privacy of the deceased, discussion of the detailed observations is nonetheless necessary because I find the medical information relevant to determine whether Mr. W. died due to coal workers' pneumoconiosis.)

Prior to summarizing the autopsy report, a review of the regulatory provisions on the requisite standard for diagnosing pneumoconiosis based on a biopsy or autopsy is helpful. The regulation at 20 C.F.R. § 718.201(a)(1) defines "clinical" pneumoconiosis as a condition:

> characterized by permanent deposition of substantial amounts of particulate matter, caused by coal dust exposure, in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis, and silicotuberculosis arising out of coal mine employment.

At the same time, because the regulatory definition of clinical pneumoconiosis requires both deposit of coal dust matter and lung tissue reaction to the deposit, an autopsy finding of

anthracotic pigmentation, standing alone, is not sufficient to establish the presence of pneumoconiosis, 20 C.F.R. § 718.202(a)(2).

Dr. Rosalie M. Uht
(DX 14)

On March 12, 2004, Dr. Uht, board certified in anatomic pathology[44] conducted a chest-only autopsy of Mr. W. According to Dr. Uht, Mr. W. had a medical history of severe pulmonary fibrosis secondary to 20 years of coal mine exposure. He was admitted to the hospital on March 9, 2004 for an acute exacerbation of his pulmonary disease and fever. Mr. W. elected for DNR/DNI status on March 11, 2004, and he passed away later that day after his respiratory failure worsened.

Upon gross examination, Mr. W. had significant clubbing and cyanosis of the fingers and toes. The serous cavities displayed extensive parietal plaques with fibrosis and thickening. Adjacent to these plaques, the pleural surface of the lungs had significant, diffuse cobble stoning with visceral pleural thickening over the upper lobes. Cut sections revealed tan-brown parenchyma with severe, diffuse fibrosis, emphysematous changes, bronchiectasis, and anthracosis. There were focal anthracotic scars in the lower lobes, adjacent to the lobar bronchi. The bronchial tree main stem was completely obstructed with mucus and severe bronchiectasis filled with mucus. The heart showed mild atherosclerosis. The left ventricular wall was 1.3cm and the right wall was 0.4cm. The right ventricle was mildly dilated. The pulmonary artery was notable for focal fatty streaking and a small amount of atherosclerotic change consistent with pulmonary hypertension. The aorta showed moderate atherosclerosis.

Under the microscope, the heart showed hypertrophic changes in the left ventricle, the right ventricle showed fatty infiltration, and the left circumflex artery showed intimal hyperplasia without atherosclerotic plaques. The lungs showed diffuse, severe interstitial fibrosis, most pronounced in the peripheral sub-pleural region. There was honeycomb-like change, with cystic dilation of air spaced lined by bronchial-type epithelium, smooth muscle hyperplasia, vascular thickening, and mucus plugging. Focal squamous metaplasia was present. Pulmonary hypertension changes were present. In the "relatively spared" regions of the lungs, the alveolar walls showed mild fibrosis with severe congestion, extensive alveolar macrophages, and development of myxoid fibrocellular organization similar to organizing pneumonia. Microscopic foci of airspaces were filled with neutriphils. Examination of the black scars seen upon gross exam showed extensive hyalinization with central necrosis, anthracosis, and grey-blue particles that were polarizable. These areas were consistent with anthracosilicotic nodules. No evidence of asbestos bodies. The visceral pleura displayed moderate thickening and fibrosis with focally-entrapped adipocytes. The parietal pleural showed moderate thickening and fibrosis, left greater than right, consistent with pleural plaques. The mediastinal lymph node displayed hyperplasia and anthracosis, with a large portion obliterated by hyalinizing fibrosis and anthracosis, consistent with a hyalinized granuloma. Dr. Uht concluded, "few anthracosilicotic nodules present centrally."

[44]I take judicial notice of Dr. Uht's board certification and have attached the certification documentation.

In determining a pulmonary diagnosis, as a preliminary matter, Dr. Uht noted that simple coal workers' pneumoconiosis is characterized by anthracotic macules and nodules, usually without clinical symptoms. Complicated coal workers' pneumoconiosis is characterized by the development of large areas of fibrosis, typically large black masses present in the upper lobes, resulting in the clinical symptoms of obstruction and restriction. For coal workers exposed to a combination of coal dust and silica, a separate syndrome of mixed dust fibrosis is frequently seen. This is characterized by patchy interstitial fibrosis forming stellate nodules with variable pigmentation. These are present diffusely in the lung parenchyma, commonly with large areas of uninvolved lung parenchyma between fibrotic zones. Interstitial pulmonary fibrosis, particularly usual interstitial pneumonia ("UIP"), is histologically characterized by a heterogeneous fibrosis with honeycomb changes separated by intervening patches of normal lung.

Dr. Uht then commented that Mr. W.'s lung histology was not typical for classic coal workers' pneumoconiosis. Instead, it was more typical for a mixed-dust fibrosis pattern with end-stage disease. Mr. W.'s recent pulmonary deterioration, his age, and the heterogeneity of the pulmonary histology raised the possibility of usual interstitial pneumonia. Although mixed dust fibrosis was "certainly" present, the possibility of usual interstitial fibrosis could not be excluded. The "terminal acute exacerbation" of Mr. W.'s lung function was probably secondary to the patchy organizing pneumonia and diffuse alveolar damage identified. These changes extensively involved the relatively spared pulmonary parenchyma. Viral infection was more likely a cause than bacteria given then lack of acute inflammation or bacterial colonization.

In Dr. Uht's opinion, Mr. W.'s cause of death was respiratory failure due to "end-stage pulmonary fibrosis (honeycomb lung)." Mr. W. also had diffuse alveolar damage (hyaline membrane disease) and patchy organizing pneumonia, bilateral pleural plaques, secondary pulmonary hypertension, and atherosclerosis.

Dr. Kyle D. Enfield
(DX 10)

Dr. Kyle D. Enfield signed Mr. W.'s death certificate. According to the physician, Mr. W.'s death on March 11, 2004 was caused by hypoxia due to pulmonary fibrosis due to pneumoconiosis.

Dr. Erika C. Crouch
(EX 1)

On June 18, 2004, Dr. Crouch, board certified in anatomic pathology, reviewed 8 slides of lung tissue from Mr. W.'s autopsy and Dr. Uht's autopsy report. Upon microscopic evaluation, the lungs showed "exudative" diffuse alveolar damage with hyaline membranes superimposed on a chronic organizing pneumonia with extensive honeycombing and associated obstructive pneumonitis. The honeycombing was predominantly subpleural and periseptal. The intervening areas of relatively normal alveoli showed scattered hyaline membranes and evidence of pneumocyte activation and proliferation. The pulmonary arteries showed evidence of hypertension consistent with the observed fibrotic lung disease. Mild multi-focal deposition of irregular black and brown particles consistent with coal dust. Although some areas of

honeycombing showed non-specific entrapment of dust, no definitive coal dust macules were observed, nor micronodules, nodules, or large lesions. "Many areas of lung fibrosis are virtually devoid of inhaled dust." A few ferruginous bodies were present, but no asbestos bodies, and no small airway changes suggestive of asbestos-related small airways disease. One slide showed a bronchopulmonary lymph node with old hyalinized granuloma and non-specific entrapment of dust particles. Two slides showed non-specific fibrous pleural plaque. No evidence of malignancy.

Dr. Crouch found coal dust deposition, but did not find evidence of pneumoconiosis or other occupational lung disease. The extensive honeycombing and fibrosis did not display a pattern secondary to coal dust inhalation or occupational asbestos exposure, but was most consistent with usual interstitial pneumonia and suggests possible idiopathic pulmonary fibrosis. The findings did not suggest asbestosis. The observed pleural plaques were non-specific, because they can result after a variety of pulmonary inflammatory reactions. The etiology of the exudative diffuse alveolar damage was unclear; possibly an exacerbation of the underlying chronic organizing pneumonia, the effect of superimposed infection, or other etiologies.

In Dr. Crouch's opinion, there was no evidence of occupational lung disease, so occupational dust exposure could not have caused an clinically significant degree of respiratory impairment or disability and could not have caused, contributed to, or hastened Mr. W.'s death.

Dr. Francis H.Y. Green
(CX 2)

On January 4, 2006, Dr. Green, board certified in anatomic pathology, reviewed Mr. W.'s medical record and reports, including Mr. W.'s living miner claim, various studies and medical reports dated October 2001 to February 2003, Dr. Cohen's medical report, Mr. W.'s death certificate, Dr. Uht's autopsy report, Dr. Crouch's report, Mr. W.'s hospitalization records, and eleven autopsy slides. Mr. W. had 20-24 years of coal mine employment of which 20 were underground.

Under the microscope, the slides of the left ventricle were normal. A section of the right ventricle showed evidence of hypertrophy. The slides of the parietal pleura showed the "classic appearance of pleural plaques." In all lung sections, the "normal architecture is severely distorted by extensive interstitial fibrosis with honeycombing." Virtually no normal tissue is seen. Thick bands of fibrosis contain varying amounts of coal mine dust, and under polarized light, considerable quantities of silica and silicates. Classic coal workers' pneumoconiosis macules or micronodules were not seen and there was no evidence of confluent fibrosis that would support a finding of PMF. The interstitial fibrosis with extensive pigmentation is "best characterized as coal workers' pneumoconiosis—interstitial fibrosis type." The tracheo-bronchial lymph nodes were largely replaced by confluent silicotic nodules, one of which was 1.5 cm in diameter. These lesions were not granulomas and showed silica and silicate under polarized light. Because these nodules were in the lymph nodes instead of the lung parenchyma, they were not diagnosed as PMF. The large airways were unremarkable and did not show chronic bronchitis, and there was no evidence of significant emphysema. The pulmonary vessels showed severe hypertensive changes characterized by increased thickness of the muscle wall and

narrowing of the lumen. There were also acute changes of diffuse alveolar injury with hyaline membranes and areas of acute inflammation indicative of acute pneumonia. There were also foci of organizing pneumonia.

Dr. Green ruled out idiopathic pulmonary fibrosis because the most common form of it has a distinctive pattern not seen in Mr. W.'s slides. The tendency of the fibrosis to be patchy and of varying onset with idiopathic pulmonary fibrosis is also consistent with asbestosis and the interstitial fibrosis of coal miners. Additionally, a report of the European Respiratory Society and the American Thoracic Society stated that known causes of interstitial fibrosis must be ruled out before making the diagnosis of idiopathic pulmonary fibrosis. Mr. W.'s occupational history and the presence of silica and silicates in his lungs mean that it is "not possible to reasonably justify diagnosing a rare disease of unknown cause."

Dr. Green concluded that Mr. W.'s pneumoconiosis was the "less common but nonetheless well-recognized variant" of coal workers' pneumoconiosis. Although it is not the most common pneumoconiosis among coal miners, it is seen in many other types of pneumoconiosis, including asbestosis, silicosis, silicate-induced pneumoconiosis, and mixed mineral dust exposures. The lungs showed considerable amounts of coal mine dust "with a relative excess" of birefringent particles consistent with silica and silicates. There were also large confluent silicotic nodules measuring greater than 1.5 cm in the lymph nodes.

Based on this review, Dr. Green diagnosed 1) hypertrophy of myocardial fibers of right ventricle, 2) mild atherosclerosis, 3) multiple pleural plaques, 4) severe end-stage interstitial fibrosis with coal mine dust, silica, and silicate pigmentation consistent with coal workers' pneumoconiosis, 5) changes consistent with severe pulmonary hypertension, 6) diffuse alveolar injury, and 7) acute and organizing pneumonia.

Mr. W.'s pulmonary history showed that he had severe end-stage pulmonary fibrosis, was severely impaired, was on home oxygen, and was placed on the lung transplant list. The pulmonary impairment began 9 or 10 years before his death with the onset of dyspnea, particularly upon exertion. Pulmonary function tests in 2001 showed evidence of restrictive lung disease and there was severe arterial hypoxemia on exercise and reduced carbon monoxide diffusing capacity. By 2003 Mr. W. had clubbing, severe restrictive disease, chest x-ray evidence of interstitial fibrosis in all zones of his lungs but most severe in the lower and middle zones, and increasing evidence of right heart enlargement seen in the echocardiogram of March 11, 2004, which is indicative of cor pulmonale. In the last years of his life, Mr. W. had several exacerbations of respiratory failure, usually precipitated by infections.

In Dr. Green's opinion, there appeared to be "unequivocal evidence" of respiratory failure due to end-stage interstitial fibrosis and that the immediate precipitating cause of death was pneumonia. Mr. Ward died of end-stage lung disease, the cause of which was pneumoconiosis due to exposure to coal mine dust during his 20-24 years of work in the coal mine industry.

Dr. Robert A.C. Cohen
(CX 1)

On July 7, 2004, Dr. Cohen, board certified in internal medicine, provided a consulting medical opinion based on Mr. W.'s 20-year coal mine work history, a cigarette smoking history of either 33 pack years[45] or 19 pack years, the medical test results of Dr. Rasmussen and Dr. Zaldivar, various tests from October 8, 2001 and January 29, 2003, an "normal" electrocardiogram from June 14, 2001,[46] Mr. W.'s death certificate, Dr. Uht's autopsy report, and Dr. Crouch's pathology review. Dr. Cohen wrote that Mr. W.'s autopsy findings "clearly show a mixed dust fibrosis pattern." Mr. W. was exposed to silica as a roof bolter. The prosector noted that the possibility of usual interstitial pneumonitis could not be ruled out. While Dr. Wiot[47] and Dr. Crouch suggested that the disease was entirely idiopathic pulmonary fibrosis, this is only appropriate when all other known causes of interstitial lung disease are excluded. It is well known that lung scarring which appears quite similar to interstitial pulmonary fibrosis can result from coal mine dust and silica dust, which is why it is mandatory to exclude such exposures when making a diagnosis. There is no basis for a finding of interstitial pulmonary fibrosis of unknown etiology where, as here, there are clear and convincing pathologic findings specific for coal dust induced lung disease.

Based on his review, Dr. Cohen found that Mr. W.'s pulmonary function tests showed a moderate to severe restrictive lung disease and a moderately severe diffusion impairment. Mr. W. also had severe gas exchange abnormalities with exercise and was on oxygen by at least 2003. The medical history indicates that Mr. W.'s 21 years of coal dust exposure was the primary cause of his pulmonary scarring, which resulted in moderate restrictive lung disease with severe diffusion impairment and severe gas exchange abnormalities with exercise. Mr. W.'s resulting respiratory impairment was a primary cause of his death.

Dr. Andrew J. Ghio
(EX 4 and EX 7)

On January 16, 2006, Dr. Ghio, board certified in internal and preventative medicine and a board certified medical examiner, reviewed Mr. W.'s medical record, including the autopsy reports of Dr. Uht and Dr. Crouch. Mr. W. was a coal miner for 20 to 25 years. He smoked cigarette for 20 years until he stopped in either 1970 or 1993. Mr. W. had a history of severe lung disease and was diagnosed with COPD. Mr. W. had interstitial fibrosis, and the complications of his lung disease included cor pulmonale and pneumonias. He was on home oxygen but walking even short distances would decrease his oxygen saturation.

Dr. Ghio noted that there was evidence of a severe injury in Mr. W.'s lungs with respiratory symptoms, abnormalities on physical exam, hypoxemia, and radiographic evidence of

[45]A pack year equals the consumption of one pack of cigarettes a day for one year.

[46]This report is not in the record.

[47]Dr. Wiot's opinion is summarized in the chest x-ray section below.

interstitial lung disease. The patient's clinical presentation "in no way resembles that of an individual with coal workers' pneumoconiosis but rather supports the pathological diagnosis of idiopathic pulmonary fibrosis." Dr. Ghio pointed to the severe symptoms that Mr. W. noted, hypoxemia, the irregular markings on the chest x-rays at Mr. W.'s lung bases, and the CT scan. Dr. Ghio also noted that respiratory failure and death are expected outcomes of idiopathic pulmonary fibrosis but not simple coal workers' pneumoconiosis.

Dr. Crouch found no coal macules under the microscope. Her description of the lung injury is not consistent with coal workers' pneumoconiosis, but is "the classical pathological presentation of idiopathic pulmonary fibrosis." Although there was another pathologist's opinion that coal workers' pneumoconiosis was present, "to discriminate between idiopathic pulmonary fibrosis and coal workers' pneumoconiosis may require an expert opinion such as Dr. Crouch's."

Dr. Ghio wrote that legal pneumoconiosis includes chronic lung diseases arising out of coal mine employment. Mr. W. suffered from idiopathic pulmonary fibrosis. There is no role for coal dust in the medical literature in causing or exacerbating idiopathic pulmonary fibrosis.

Based on his review, Dr. Ghio opined that coal workers' pneumoconiosis did not have any significant effect on Mr. W.'s death because he did not have coal workers' pneumoconiosis. If Mr. W. was found to have coal workers' pneumoconiosis, Dr. Ghio said that his opinion regarding the cause of Mr. W.'s death would not change from death due to idiopathic pulmonary fibrosis.

At a deposition on May 19, 2006, Dr. Ghio noted that Mr. W.'s 20 to 25 years in the coal mines was a sufficient amount of time to cause a dust-induced lung disease in a susceptible individual. Mr. W.'s chest x-rays were "very consistent" with interstitial lung disease, specifically idiopathic pulmonary fibrosis, and more specifically usual interstitial pneumonitis. Coal workers' pneumoconiosis would involve superior positions of the lung, with rounded or small opacities involving both lungs. Usual interstitial pneumonitis has irregular opacities in the lower portions of the lung, and that is what Mr. W.'s chest x-rays showed. Pulmonary function tests can show a restrictive disease and a loss of diffusing capacity. This can occur with any interstitial lung disease, including coal workers' pneumoconiosis and usual interstitial pneumonitis. It is rather unusual in simple coal workers' pneumoconiosis, but it is more common in usual interstitial pneumonitis. With regard to the arterial blood gas studies, the severity of the oxygen decrement in Mr. W. is "very rarely" seen with coal workers' pneumoconiosis, but would be frequently seen with usual interstitial pneumonitis. Based on these two types of tests, this is either an extremely unusual case of coal workers' pneumoconiosis or it is what would be frequently seen in a patient with usual interstitial pneumonitis.

Dr. Ghio agreed that a miner who worked underground for 20 to 25 years would probably have exposure to silica. Silica is more fibrogenic than coal dust, and it may also have a toxic effect on the lung. Encased in fibrosis, that toxic effect may remain. If the profusion is severe enough, the scarring process of coal workers' pneumoconiosis can result in a restrictive and dilatory effect, even though that is more common with complicated coal workers' pneumoconiosis. Coal workers' pneumoconiosis could also result in inspiratory crackles, though

it is unusual. Coal workers' pneumoconiosis can result in a significant drop in arterial oxygen, but it is very rare. Clubbing and cyanosis can accompany a reduced blood oxygen level. As someone reaches the upper levels of coal workers' pneumoconiosis, the speed of the disease can quicken. The preponderance of the data in Mr. W.'s case supports a diagnosis of COPD, but there is no evidence of an obstruction. If the physicians who diagnosed COPD were correct, it would be hard to rule out the contribution of coal mine dust to that disease. However, Mr. W. did not have evidence of an obstruction, and Dr. Ghio had never seen such severe O2 reductions.

Dr. Ghio also reviewed Dr. Green's report, but confirmed that his overall opinion remained consistent with Dr. Crouch's interpretation. Dr. Green's finding of coal workers' pneumoconiosis was not supported because he did not find macules. Under the microscope, usual interstitial pneumonitis looks different from coal workers' pneumoconiosis; usual interstitial pneumonitis does not display macules, it shows honeycombing more frequently, and the extent of the injury is greater. Those three elements were true in Mr. W.'s lungs. The presence of coal dust in a slide does not translate into a diagnosis of coal workers' pneumoconiosis; a fibrotic reaction is needed. Instead, Dr. Green's report was consistent with usual interstitial pneumonitis. Dr. Ghio did not agree with Dr. Cohen's findings, and that report did not change Dr. Ghio's opinions.

Lung disease can be multifactorial, but the pathology is not consistent with coal workers' pneumoconiosis in this case. The American Thoracic Society says that before a diagnosis of idiopathic pulmonary fibrosis, a physician must rule out other exposures that could cause a fibrosis. There are typical and atypical varieties of coal workers' pneumoconiosis, but Mr. W.'s presentation would be so atypical as to make it "close to zero." Still, Dr. Ghio agreed that he could not rule it out. Dr. Ghio also agreed that pathology and autopsy evidence is the "gold standard" in diagnosing the presence or absence of pneumoconiosis.

Dr. Ghio stated that Mr. W.'s death was secondary to respiratory failure due to pulmonary fibrosis. He also stated that Mr. W. would have died from the same respiratory failure if he had never stepped into a coal mine.

Dr. Thomas M. Jarboe
(EX 5 and EX 6)

On February 1, 2006, Dr. Jarboe, board certified in internal medicine and a board certified medical examiner, reviewed Mr. W.'s employment history and medical record, including Mr. W.'s death certificate, Dr. Uht's autopsy report, and Dr. Crouch's pathology consultation. Based on his review, Dr. Jarboe concluded that Mr. W. suffered from "idiopathic pulmonary fibrosis (usual interstitial pneumonitis)" and not coal workers' pneumoconiosis. Mr. W. left the mines in 1992, but there are no medical records that show any significant pulmonary impairment at that time. Later in 2003, Dr. Robbins noted that Mr. W. noted worsening decline over the prior 8 years. "The available records frame an illness which spanned a relatively short period of time." Mr. W.'s illness took a very rapid course over three to four years. Between October 2001 and three years later, Mr. W.'s lung function deteriorated "extremely" rapidly. The CT scan showed a predominantly basilar disease and honeycombing in the subpleural location. This is the classical distribution for idiopathic pulmonary fibrosis. It would be unusual

for coal workers' pneumoconiosis to be more predominant in the lung bases. The clinical findings also support idiopathic pulmonary fibrosis, in the form of crackles at the bases and clubbing of the digits, which are not seen with coal workers' pneumoconiosis. Diffuse alveolar damage was also present, and that occurs in the accelerated phase of idiopathic pulmonary fibrosis.

In terms of autopsy evidence, Dr. Jarboe observed that although Dr. Uht stated that the histologic findings were more consistent with mixed dust fibrosis with end stage disease, she did not describe the findings to support that statement. Her reference to a scar in the lower lung zone is "entirely nonspecific," and it is unclear that it is a lesion of coal workers' pneumoconiosis. Dr. Crouch commented that much of the black pigment in the lung was trapped in scarred areas caused by the interstitial fibrosis. Dr. Uht did not document that any of the fibrotic areas, except for the black scar, contained silica. Additionally, Dr. Uht does not list coal workers' pneumoconiosis as a cause of death.

Coal workers' pneumoconiosis did not contribute to or hasten Mr. W.'s death. There was no pathological evidence that Mr. W. had that disease. Mr. W. had idiopathic fibrosis which caused rapid deterioration of his respiratory status with the development of severe hypoxemic respiratory failure and death. There was no evidence that coal workers' pneumoconiosis contributed to his death in any way. It is possible that that pigmented scars are indicative of coal workers' pneumoconiosis, but these scars did not involve a large area of lung volume and were adjacent to the lobar bronchi. Based on that description, the scarring would not have been sufficient to have impaired Mr. W.'s function. Dr. Crouch did not describe lesions that would have contributed to impairment or death.

At a deposition on May 18, 2006, Dr. Jarboe noted that he reviewed the reports of Dr. Green and Dr. Cohen. Dr. Jarboe stated that those reports did not change his earlier assessment. He noted that both doctors reported that a finding of idiopathic pulmonary fibrosis cannot be made if there is any environmental exposure, but he disagreed. First, he was not aware of any study linking coal workers' pneumoconiosis to idiopathic pulmonary fibrosis. Second, it is illogical to say that any environmental exposure excludes a diagnosis of idiopathic pulmonary fibrosis. If a man worked in the coal mines in his 20s and then developed idiopathic pulmonary fibrosis in his 70s, it is untenable to exclude a diagnosis of idiopathic pulmonary fibrosis based on that environmental exposure. Dr. Jarboe agreed that exclusion of other known causes of pulmonary fibrosis is a requirement before diagnosing idiopathic pulmonary fibrosis, but also stated that it is "an extreme interpretation of that statement to say that other environmental causes must be ruled out before you can make the diagnosis." Coal dust has been shown to cause fibrosis, but it has not been shown to cause the classical histologic picture of idiopathic pulmonary fibrosis. Dr. Jarboe said he "simply can't go along with" the position that some coal mine work means a physician cannot say he has idiopathic pulmonary fibrosis. Dr. Jarboe conceded that whether Dr. Uht's autopsy report was "artful" or not on the issue of her mixed dust fibrosis finding, she did include it as a final opinion. Dr. Jarboe stated that he would rely very heavily on reports from either Dr. Green or Dr. Crouch. He said that if Dr. Crouch's CV lacked specific work in black lung research it "might" suggest that her specialty in pathology is not as specific as Dr. Green's.

Even if Mr. W. was diagnosed with coal workers' pneumoconiosis, mild simple coal workers' pneumoconiosis would not have caused the pathological changes noted in the chest x-rays, nor the rapidly progressive pulmonary insufficiency and death in this case. Dr. Jarboe clarified that he was using "mild simple coal workers' pneumoconiosis" to describe Dr. Green's findings of dust macules.

Idiopathic fibrosis is not very rare, Dr. Jarboe sees a case of it several times a year, but it is not a common condition like asthma or "garden-variety" COPD. Dr. Jarboe did not see evidence for a diagnosis of COPD, based on Mr. W.'s pulmonary function test in 2001. His 2003 test showed a 90% FEV1/FVC ratio, which is "highly characteristic" of idiopathic pulmonary fibrosis because the patient gets a "small stuff lung that empties rapidly." The ratio tends to go up over time, which is what happened with Mr. W. between 2001 and 2003. For coal workers' pneumoconiosis, the ratio would be closer to 75% or 78%. If Mr. W. had shortness of breath for 20 years, it could indicate that he had both pneumoconiosis and idiopathic pulmonary fibrosis.

Dr. Jarboe agreed that medical diagnosis works by moving from the most likely diagnosis to less and less likely diagnoses when making differential diagnoses. He also agreed that the statistical incidence of coal workers' pneumoconiosis is greater than that of idiopathic pulmonary fibrosis. When asked if there was anything in Mr. W.'s pulmonary function testing that could rule out coal workers' pneumoconiosis, Dr. Jarboe pointed to the rapid deterioration of Mr. W.'s forced vital capacity over a few years. When asked if idiopathic fibrosis and coal workers' pneumoconiosis, in combination, could cause such a drop, Dr. Jarboe agreed, but said that there was no evidence of a huge amount of coal dust in Mr. W.'s lungs, which didn't argue in favor of that causing the rapid deterioration. Instead, idiopathic fibrosis was the "most likely" cause of that rapid deterioration.

Dr. Jarboe agreed that Mr. W. had severe interstitial fibrosis, with crackles on physical examination, severe restrictive lung disease, reduced diffusing capacity, clubbing of the fingernails, and a reduced blood oxygen level. Clubbing of the digits and "velcro rales" are "very uncommon" but Mr. W.'s other symptoms may be seen with coal workers' pneumoconiosis. If a part of Mr. W.'s lung disease was due to the inhalation of coal mine dust, Dr. Jarboe said that Mr. W. could have legal pneumoconiosis, but Dr. Jarboe said it would be hard to separate that out. Additionally, clubbing and crackles do not rule out pneumoconiosis, they could come from some other cause even if pneumoconiosis was present. Diffuse alveolar damage, although not a sign of coal workers' pneumoconiosis, could also be present in addition to coal workers' pneumoconiosis. One of the most common reasons to get a lung transplant is the presence of idiopathic fibrosis. If the majority of Mr. W.'s fibrosis was found to come from coal workers' pneumoconiosis and not interstitial pulmonary fibrosis, then it would be fair to say that his death was caused and hastened by coal mine employment.

Complicated Pneumoconiosis

The regulation, in part, at 20 C.F.R. § 718.304, provides that if a claimant is able to establish the presence of complicated pneumoconiosis, then an irrebuttable presumption of total disability and death due to pneumoconiosis is established.

In the Black Lung Benefits Act, 30 U.S.C. 921(c)(3)(A) and (C), as implemented by 20 C.F.R. § 718.304(a), Congress determined that if a miner suffered from a chronic dust disease of the lung which "when diagnosed by chest X-ray, yields one or more large opacities (greater than one centimeter in diameter) and would be classified in category A, B, or C," there shall be an irrebuttable presumption that his death was due to pneumoconiosis.[48] This type of large opacity is called "complicated pneumoconiosis." The statute and regulation also permit complicated pneumoconiosis to be established by either the presence of massive fibrosis in biopsy and autopsy evidence or other means which would be expected to produce equivalent results in chest x-rays or biopsy/autopsy evidence. 30 U.S.C. 921(c)(3)(B) and (C) and 20 C.F.R. §§ 718.304(b) and (c). Additionally, a diagnosis of progressive massive fibrosis is consistent with a finding of complicated pneumoconiosis. The Supreme Court recognized complicated pneumoconiosis as "involv[ing] progressive massive fibrosis as a complex reaction to dust and other factors." *Usery v. Turner Elkhorn Mining Co*., 428 U.S. 1, 7 (1976). Moreover, the Fourth Circuit commented that complicated pneumoconiosis is also known "by its more dauntingly descriptive name, 'progressive massive fibrosis'." *Lisa Lee Mines v. Director, OWCP*, 86 F.3d 1358, 1359 (4th Cir. 1996).

According to the Fourth Circuit in *Eastern Associated Coal Corp. v. Director, OWCP [Scarbro]*, 220 F.3d 250 (4th Cir. 2000), the existence of complicated pneumoconiosis is established by "congressionally defined criteria." As a result, the statute's definition of complicated pneumoconiosis as radiographic evidence of one or more large opacities categorized as size A, B, or C represents the most objective measure of the condition. This sets the benchmark by which other methods for proving complicated pneumoconiosis are measured, as described in 30 U.S.C. 921(c)(3)(B) and (C). *Scarbro,* 220 F.3d at 256. In other words, whether a massive lesion or other diagnostic results represent complicated pneumoconiosis under 30 U.S.C. 921(c)(3)(B) and (C) requires an equivalency evaluation with the x-ray criteria set forth in 30 U.S.C. 921(c)(3)(A).[49]

With these principles in mind, I first note that no radiographic evidence of a large pulmonary opacity exists. Next, in terms of other evidence, during his review of the autopsy slides, Dr. Green observed silica nodules in the bronchial lymph nodes, measuring up to 1.5 centimeters.[50] However, while such silica nodules support a finding of pneumoconiosis, no physician in the record rendered the requisite equivalency determination that the 1.5 cm biopsy nodule would appear as an opacity greater than one centimeter on a chest x-ray. In the absence

[48]On the standard ILO chest x-ray classification worksheet, Form CM 933, large opacities are characterized by three sizes, identified by letters. Category A indicates the presence of a large opacity having a diameter greater than 10 mm (one centimeter) but not more than 50 mm; or several large opacities, each greater than 10 mm but the diameter of the aggregate does not exceed 50 mm. Category B means an opacity, or opacities "larger or more numerous than Category A" whose combined area does not exceed the equivalent of the right upper zone of the lung. Category C represents one or more large opacities whose combined area exceeds the equivalent of the right upper zone.

[49]*See also* 20 C.F.R. §§ 718.304(b) and (c).

[50]In *Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpub.), the BRB noted that "anthracosis found in lymph nodes may be sufficient to establish the existence of pneumoconiosis."

of that equivalency finding, Mrs. W. is unable to establish the presence of complicated pneumoconiosis.

Death Due to Pneumoconiosis

In considering the numerous medical opinions, I first note the obvious that the admitted treatment records and pulmonary evaluations pre-date Mr. W.'s death and provide no direct evidence on the cause of his death. However, that medical evidence remains important because it establishes significant aspects of Mr. W.'s pulmonary health in his final years.

Seven physicians presented conflicting opinions on the cause of Mr. W.'s death. Due to the conflict in the medical reports, I must evaluate the relative probative value of the conflicting evidence in terms of documentation and reasoning. As to the first factor, a physician's medical opinion is likely to be more comprehensive and probative if it is based on extensive objective medical documentation such as radiographic tests and physical examinations. *Hoffman v. B & G Construction Co.*, 8 B.L.R. 1-65 (1985). In other words, a doctor who considers an array of medical documentation that is both long (involving comprehensive testing) and deep (includes both the most recent medical information and past medical tests) is in a better position to present a more probative assessment than the physician who bases a diagnosis on a test or two and one encounter.

The second factor involves an evaluation of the connections a physician makes based on the documentation before him or her. A doctor's reasoning that is both supported by objective medical tests and consistent with all the documentation in the record, is entitled to greater probative weight. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19 (1987). Additionally, to be considered well reasoned, the physician's conclusion must be stated without equivocation or vagueness. *Justice v. Island Creek Coal Co.*, 11 B.L.R. 1-91 (1988).

With these probative factors in mind, I first find two assessments clearly have diminished probative value. First, on the death certificate, Dr. Enfield attributed Mr. W's death to hypoxia due to pulmonary fibrosis due to pneumoconiosis. Without any accompanying explanation for his conclusions, Dr. Enfield's conclusion loses probative value for insufficient reasoning. Second, as the underlying basis for her conclusion that pneumoconiosis did not cause, contribute to, or hasten Mr. W.'s death, Dr. Crouch relied solely on her pathological assessment that Mr. W. did not coal workers' pneumoconiosis. However, the law of the case dictates that Mr. W. had coal workers' pneumoconiosis. Consequently, Dr. Crouch's opinion has little probative value in assessing the role the coal workers' pneumoconiosis in Mr. W.'s lungs may have played in his death.

Turning to the remaining five medical opinions, in light of the extensive nature of their medical reports, a brief summarization of their assessments facilitates the probative value determination regarding their respective cause of death analysis.

Dr. Uht

Dr. Uht rendered three principal pulmonary findings. First, upon gross and microscopic examination, Dr. Uht observed severe, diffuse and honeycombed interstitial fibrosis. Although Mr. W.'s lung tissue did not contain coal dust macules associated with classic coal workers' pneumoconiosis, his diffuse, patchy fibrosis was consistent with another syndrome of "mixed dust" fibrosis associated with exposure to coal dust and silica. In addition to the mixed dust component, in light of the rapid decline in Mr. W.'s respiratory capacity, and the heterogeneous nature of the fibrosis, a possibility of superimposed UIP existed. Second, in relatively spare areas of lungs, Dr. Uht found evidence of organizing pneumonia. Third, after noting focal anthracotic scars in the lower lobes during the gross assessment, Dr. Uht identified under the microscope with polarized light particles that were consistent with anthracosilicotic nodules, which legally represents a diagnosis of coal workers' pneumoconiosis.[51] She characterized the extent of the anthracosilicotic nodules as "few."

Based on the three pulmonary findings, Dr. Uht opined that the organizing pneumonia exacerbated Mr. W. 's pulmonary disease. As a result, he suffered a respiratory death due to end-stage pulmonary fibrosis and organizing pneumonia.

In assessing probative value of Dr. Uht's opinion, I first note she did not indicate that the "few" anthracosilicotic nodules played any role on Mr. W.'s death. Additionally, Dr. Uht reported that Mr. W. did not have classical coal workers' pneumoconiosis. However, Dr. Uht also concluded that the abnormal fibrotic lung tissue that she observed during the autopsy and microscopic evaluation, was in part "mixed dust" fibrosis attributable to Mr. W.'s exposure to coal dust and silica. Although coal mine dust pigmentation standing alone is insufficient for a diagnosis of pneumoconiosis, Dr. Uht's finding of coal dust and silica deposits associated with fibrosis represents a finding of coal workers' pneumoconiosis. Since Dr. Uht further concluded that the presence of the mixed dust fibrosis was the disease process exacerbated by Mr. W.'s pneumonia, I find Dr. Uht's documented and reasoned medical opinion supports a finding that the end-stage pulmonary fibrosis, which included coal workers' pneumoconiosis, contributed to Mr. W.'s death.

Dr. Green

After reviewing Dr. Uht's gross autopsy findings and microscopically examining the lung tissue slides, Dr. Green rendered two primary pulmonary diagnosis. First, he observed extensive interstitial fibrosis with honeycombing. Although no classic coal workers' pneumoconiosis macules or micronodules were present, Dr. Green observed thick bands of fibrotic lung tissue containing varying amounts of coal mine dust and "considerable quantities" and "relative excess" of silica and silicates. Additionally, large confluent silicotic nodules were present in the pulmonary lymph nodes. In light of these observations, Dr. Green diagnosed the interstitial fibrosis with extensive pigmentation as "coal workers' pneumoconiosis – interstitial fibrosis type." He explained that while Mr. W.'s coal workers' pneumoconiosis was not the most common type, it was consistent with pneumoconiosis in coal miners with mixed dust exposures. Additionally, based on an inconsistent pattern on the lung tissue samples, Dr. Green ruled out

[51] *See* 20 C.F.R. § 718.201(a)(1).

idiopathic pulmonary fibrosis. Second, Mr. W.'s lung contained areas of acute inflammation associated with active organizing pneumonia.

Based his assessment, Dr. Green concluded that Mr. W. had end-stage interstitial fibrosis caused by pneumoconiosis that arose due to his coal mine dust exposure. When pneumonia developed, it exacerbated the severe pulmonary disease causing respiratory failure and death.

Integrating Mr. W.'s occupational dust exposure, clinical presentation, and autopsy/biopsy evidence, Dr. Green presented a well documented and reasoned medical opinion that Mr. W.'s respiratory failure was caused by end-stage interstitial fibrosis due to coal mine dust-related pneumoconiosis and precipitated by pneumonia.

Dr. Cohen

Following his review of Mr. W.'s employment history, medical record, pulmonary examinations, and the autopsy evidence, Dr. Cohen concluded Mr. W. had mixed dust fibrosis attributable to his coal mine employment. Noting the conflicting assessments by Dr. Crouch and Dr. Uht, having considered the possibility of idiopathic pulmonary fibrosis, and emphasizing the absence of any other occupational dust exposure, Dr. Cohen observed that Mr. W.'s work as a roof bolter would have exposed him to silica when drilling the hard rock top. As a result, Dr. Cohen concluded Mr. W.'s lung scarring was consistent with the interstitial pulmonary fibrosis that results from exposure to coal mine dust and silica. He also noted that Mr. W.'s clinical presentation included a severe respiratory lung disease, moderately severe diffuse impairment and severe gas exchange abnormalities attributable to his coal mine dust related pulmonary scarring. Mr. W.'s significant exposure to coal dust and silica caused his severe respiratory impairment. In turn, Mr. W.'s pulmonary impairment was a primary cause of his death.

Having conducted an extensive documentary review, Dr. Cohen presented a well documented assessment. Similarly, his analysis about and conclusion that Mr. W. had mixed dust pulmonary fibrosis rather than idiopathic fibrosis that caused his death are reasoned.

Dr. Ghio

Based upon Mr. W.'s clinical presentation, Dr. Ghio opined that his symptoms were more consistent with idiopathic pulmonary fibrosis rather than coal workers' pneumoconiosis. The presence of idiopathic fibrosis was confirmed by Dr. Crouch's pathology findings. Although other pathologists diagnosed pneumoconiosis, Dr. Ghio relied on Dr. Crouch's assessment based on her expertise and the absence of any coal workers' macules in any of the pathology assessments. In light of Dr. Crouch's pathology findings, Dr. Ghio opined that even if Mr. W. had coal workers' pneumoconiosis, his death was nonetheless due to idiopathic pulmonary fibrosis.

Since Mr. W.'s pathology was not consistent with coal workers' pneumoconiosis, Dr. Ghio opined Mr. W.'s death was secondary to respiratory failure due to idiopathic pulmonary fibrosis unrelated to his coal mine employment.

Although Dr. Ghio presented an extensive analysis, his opinion has diminished probative value for a reasoning deficiency and a documentation issue. In terms of reasoning, Dr. Ghio believed Mr. W. did not have coal workers' pneumoconiosis due in part to the absence of biopsy evidence of coal dust macules. In reaching that conclusion, Dr. Ghio did not address whether the extensive pulmonary fibrosis associated with significant coal dust and silica deposit could also represent pneumoconiosis.

Documentation-wise, Dr. Ghio's reliance on Dr. Crouch's pathology report as his supporting documentation is misplaced for three reasons. First, since two of the three board certified pathologists found the presence of pneumoconiosis, Dr. Crouch's report represents the minority opinion. Second, in favoring Dr. Crouch's report, Dr. Ghio referenced her expertise. Yet, the record does not establish that Dr. Crouch had superior credentials or expertise to support favoring her assessment over the evaluations of Dr. Uht and Dr. Green. Third, in choosing Dr. Crouch's report, Dr. Ghio did not address a significant observations by Dr. Uht and Dr. Green that lead to their findings supportive of coal workers' pneumoconiosis. When Dr. Uht and Dr. Green applied polarized light, they identified significant silica and silicate particles in the pulmonary fibrosis. In contrast, Dr. Crouch simply referenced "a few ferruginous bodies." This biopsy evidence of extensive silica is a significant factor since Dr. Ghio acknowledged that Mr. W.'s long employment underground would probably have exposed him to silica, which he stated is more fibrogenic than coal mine dust and has a toxic effect on the lungs.

Dr. Jarboe

Upon evaluation of the clinical presentation and the diverse pathology reports, Dr. Jarboe concluded that Mr. W. had idiopathic pulmonary fibrosis which caused his rapid respiratory decline and death. Dr. Jarboe noted the absence of any significant coal dust related lesions in the pathology reports. While the pathology report of Dr. Green was reliable, Dr. Jarboe did not agree that the extensive pulmonary fibrosis was related to coal mine dust exposure. Instead, coal workers' pneumoconiosis nodules were located in only a small area of the lungs. Consequently, even if Mr. W. had coal workers' pneumoconiosis, its presence was too mild to have been a factor in his death.

In terms of probative value, Dr. Jarboe presented a documented assessment that coal workers' pneumoconiosis did not cause, contribute to or hasten Mr. W.'s death. In addition to an extensive medical record review, and an in-depth discussion on idiopathic pulmonary fibrosis, Dr. Jarboe also recognized the conflicting pathology reports. Although he faulted Dr. Uht's report for lack of specificity, Dr. Jarboe considered the other pathology reports which he considered reliable. And, while he did not completely explain how he reconciled the conflicting assessments by Dr. Crouch and Dr. Green, Dr. Jarboe presented a reasoned assessment that the identified coal workers' lesions in Dr. Green's report were insufficient to affect Mr. W.'s death.

Summary

For various reasoning and documentation shortfalls, the medical opinions of Dr. Enfield, Dr. Crouch, and Dr. Ghio have diminished probative value. Although Dr. Jarboe's assessment that idiopathic pulmonary fibrosis caused Mr. W.'s death and coal workers' pneumoconiosis was

too mild to have been a factor is documented and reasoned, the contrary consensus by Dr. Uht, Dr. Green, and Dr. Cohen represents the preponderance of the medical opinion on the cause of death. Based on the conclusions of Dr. Uht, Dr. Green, and Dr. Cohen that Mr. W.'s end-stage pulmonary fibrosis was mixed dust coal workers' pneumoconiosis, I find that coal workers' pneumoconiosis caused Mr. W.'s death.

CONCLUSION

Mrs. W. has shown that she is an eligible survivor. Through nonmutual, offensive collateral estoppel, Mrs. W. has established that her husband Mr. W. had coal workers' pneumoconiosis. Finally, upon evaluation of the entire record, I find that the preponderance of the probative medical opinion establishes that Mr. W.'s death was due to coal workers' pneumoconiosis. Accordingly, Mrs. W.'s claim for survivor benefits must be granted.

DATE OF ENTITLEMENT

In the case of a coal miner who died due to pneumoconiosis, benefits to the survivor are payable beginning the month the coal miner died. 20 C.F.R. § 725.503(c). Because Mr. W. passed away on March 11, 2004, Mrs. W.'s benefits are payable beginning March 1, 2004.

ATTORNEY FEES

Counsel for the Claimant has 30 days from receipt of this decision to submit an additional application for attorney fees related to this case in accordance with 20 C.F.R. §§ 725.365, 725.366. With the application, counsel must attach a document showing service of the fee application upon all parties, including the Claimant. The other parties have 15 days from receipt of the fee application to file an objection to the request. Absent an approved application, no fee may be charged for representation services associated with the claim.

ORDER

The claim of Mrs. W.W., widow of Mr. J.R.W., for survivor benefits under the Act is **GRANTED**. CONTINENTAL MINING CO. is ordered to pay Mrs. W. all survivor benefits to which she is entitled under the Act and Regulations. Benefits shall commence March 1, 2004.

SO ORDERED:

A

RICHARD T. STANSELL-GAMM
Administrative Law Judge

Date Signed: March 26, 2007
Washington, DC

NOTICE OF APPEAL RIGHTS: If you are dissatisfied with the administrative law judge's decision, you may file an appeal with the Benefits Review Board ("Board"). To be timely, your appeal must be filed with the Board within thirty (30) days from the date on which the

administrative law judge's decision is filed with the district director's office. See 20 C.F.R. §§ 725.458 and 725.459. The address of the Board is: Benefits Review Board, U.S. Department of Labor, P.O. Box 37601, Washington, DC 20013-7601. Your appeal is considered filed on the date it is received in the Office of the Clerk of the Board, unless the appeal is sent by mail and the Board determines that the U.S. Postal Service postmark, or other reliable evidence establishing the mailing date, may be used. See 20 C.F.R. § 802.207. Once an appeal is filed, all inquiries and correspondence should be directed to the Board.

After receipt of an appeal, the Board will issue a notice to all parties acknowledging receipt of the appeal and advising them as to any further action needed.

At the time you file an appeal with the Board, you must also send a copy of the appeal letter to Allen Feldman, Associate Solicitor, Black Lung and Longshore Legal Services, U.S. Department of Labor, 200 Constitution Ave., NW, Room N-2117, Washington, DC 20210. See 20 C.F.R. § 725.481.

If an appeal is not timely filed with the Board, the administrative law judge's decision becomes the final order of the Secretary of Labor pursuant to 20 C.F.R. § 725.479(a).

Attachment No. 1

American Board of Medical Specialties
Certification:

Rosalie M. Uht, MD

Certified by The American Board of Pathology in:

Anatomic Pathology

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